CASE REPORT

Dual-site right ventricular and left ventricular pacing in a patient with left ventricular systolic dysfunction and atrial fibrillation using a standard CRT-D device

David Chase [a,*], Vipin Kumar [a], Amit Hooda [a]

[a] Christian Medical College Hospital, Vellore, Tamilnadu 632004

[a] India

In patients undergoing cardiac resynchronization therapy with defibrillator (CRT-D) implantation for left ventricular systolic dysfunction (LVSD) accompanied by permanent atrial fibrillation (AF), generally, the unused atrial port is plugged at device implantation. We describe an alternative use for the atrial-port in this case report.

A 43 year old gentleman with LVSD due to left ventricular non-compaction (LVNC) and AF of unknown duration underwent a CRT-D implantation after optimization of cardiac failure treatment. The atrial-port which would otherwise have been plugged was connected to a high right ventricular septal (RVS) pacing-lead and the shock-lead was positioned at the right ventricular apex (RVA). This approach permitted modified cardiac resynchronization in a high RVS to left ventricular (LV) and RVA pacing sequence using the high RVS and LV pacing combined with a shock vector including the RV apex. A standard CRT-D device with a minimum programmable A–V delay of 30 ms (technically RVS to LV delay in the 'DDD' pacing mode) was used. The device was programmed to a 'DDD' pacing mode (sequential multi-site ventricular pacing with some programmability). The mode switch operation was programmed 'OFF' since atrial sensing is unavailable. Device-delivered shocks did not cardiovert the patient back to sinus rhythm suggesting that the AF was permanent (no prior cardioversion attempts were made on the presumption that the chances of maintaining sinus rhythm, given the underlying cardiac condition, were low). Subsequently, the patient required radio-frequency ablation of the atrio-ventricular node for conducted AF. Symptomatic, echocardiographic and radiological improvement preceded atrio-ventricular node ablation.

Conclusion: **Amongst AF patients with permanent AF undergoing CRT-D implantation, those patients who are likely to have the CRT-D device atrial-ports plugged could benefit from having both the options of (i) a RVA shock vector as well as (ii) a high RVS-pacing feasible, by utilizing the atrial-port of a conventional CRTD device for a RVS pacing lead, should a RVA shock-lead position be preferred. New device programming algorithms will be necessary to make patient-customized programming in this lead configuration flexible, more useful clinically and easy.**

© 2013 King Saud University. Production and hosting by Elsevier B.V. All rights reserved.

Received 13 October 2012; revised 6 January 2013; accepted 12 January 2013.
Available online 26 January 2013

Disclosure: Author has nothing to disclose with regard to commercial support.

* Corresponding author. Address: Department of Cardiac Pacing and Electrophysiology, (Cardiology Unit IV), Christian Medical College Hospital, Vellore, Tamilnadu 632004, India. Tel.: +91 416 2283572; fax: +91 4162232035.
E-mail addresses: davsuchi@gmail.com (D. Chase), docvipin2005@gmail.com (V. Kumar), amithooda@rediffmail.com (A. Hooda).

1016–7315 © 2013 King Saud University.
Production and hosting by Elsevier B.V. All rights reserved.

Peer review under responsibility of King Saud University.
URL: www.ksu.edu.sa
http://dx.doi.org/10.1016/j.jsha.2013.01.003

P.O. Box 2925 Riyadh – 11461KSA
Tel: +966 1 2520088 ext 40151
Fax: +966 1 2520718
Email: sha@sha.org.sa
URL: www.sha.org.sa

Production and hosting by Elsevier

CASE REPORT

Keywords: Cardiac resynchronization therapy with defibrillator (CRT-D), Left ventricular systolic dysfunction (LVSD), Atrial fibrillation (AF), Left ventricular non-compaction (LVNC), High right ventricular septum (high RVS), Right ventricular apex (RVA), Left ventricle (LV), Atrio-ventricular node (A-VN), Radio-frequency ablation (RFA)

Introduction

The benefits of medical therapy followed by cardiac resynchronization therapy (CRT) in patients who qualify for such treatment are well known [1,2]. In patients with left ventricular systolic dysfunction and permanent atrial fibrillation, CRT-D implant procedure usually involves plugging the atrial-port, unless it is preferred to retain the atrial sensing option. The implanting physician's personal preference or compelling patient's anatomy or another such factor would determine whether the RV shock-lead is positioned at the RVA or in the high RVS. The debate as to which RV pacing site is superior is far from over. Further, many operators still prefer the RVA site for device-delivered shocks and at times others whose routine practice regarding positioning the RV shock-lead involves the RVS also end up having to change to a RVA site if a high defibrillation threshold is encountered. In any case, RV shock-lead location limits options in the sense that the same site is going to be used for pacing and for device-delivered shocks. Cardiac resynchronization therapy requires predominant multi-site ventricular pacing (minimal, if not no ventricular sensing, at times with the aid of atrio-ventricular node ablation) for maximal benefits. Flexibility in device programming for allowing multiple options in various combinations of the LV, RVS and RVA is presently unavailable.

In this case report, we describe a modification of the conventional CRT-D implant procedure with a view to utilize the atrial port for RVS pacing with a standard bipolar active fixation pacing lead and utilizing the shock lead in the RVA for device-delivered shock which includes the apical region of the heart. This might become important in a patient with significant cardiomegaly and a high defibrillation threshold from the RVS. Unfortunately, when used in this manner, the lowest programmable A*–V delay (* the atrial port is actually connected to the RVS, making it a RVS-V delay) which is 30 ms in some devices, mandates that RVS pacing occurs first, followed by left ventricular (LV) or RVA depending on how the other ventricular leads and V–V delay ('non RVS' V-leads) are programmed. In addition, mode-switch operation would need to be turned 'OFF' as there is no atrial sensing and for the same reason, 1:1 V–A relationship algorithms would become meaningless in device therapy programming. We presumed that a 30 ms early septal pacing would not have a significant impact (markedly prolonged baseline QRS duration) and in the event of not seeing a significant response in terms of clinical improvement, we were prepared to fall back on to a standard bi-ventricular 'VVIR' mode.

Clinical findings and investigation reports

A 43 year old gentleman presented with progressive dyspnoea on exertion (New York Heart Association symptom class III at admission) for five years, pedal oedema/paroxysmal nocturnal dyspnoea for one month in spite of medical treatment given at his primary hospital and was referred for refractory symptoms. There was no significant history suggesting alcohol or substance use. The jugular venous pressure was elevated and pedal oedema was present. The pulse was irregularly irregular with a rate of 74 beats per minute and the blood pressure was 110/80 mm Hg. A pansystolic murmur could be heard at the apex. There was no other significant clinical finding at examination.

Routine 12 lead ECG confirmed the presence of atrial fibrillation and left bundle branch block with a QRS duration of 174 ms (see Fig. 1). The chest X-ray revealed cardiomegaly (cardio-thoracic ratio of 0.75) and evidence of pulmonary venous congestion. Mild pre-renal form of uraemia was present but otherwise clinical pathology and biochemistry testing did not reveal any other abnormality. Thyroid function and liver function was normal. Echocardiography revealed left ventricular non-compaction (confirmed later by magnetic resonance imaging), dilated left ventricle (LVIDD/IDS – 67/58 mm) with global hypokinesia, severe left ventricular systolic dysfunction (LV ejection fraction by Simpsons method – 22%), moderate mitral and tricuspid regurgitation and evidence of pulmonary hypertension. The pulmonary arterial pressure at cardiac catheterization was 59/31 mmHg with a mean of 40 mmHg and the left ventricular end-diastolic pressure was elevated at 25 mmHg. Coronary angiography did not reveal any significant flow limiting disease.

Treatment details

The patient's therapy with Carvedilol/Ramipril/Diuretics/Spirono lactone/Digoxin/oral anticoagu-

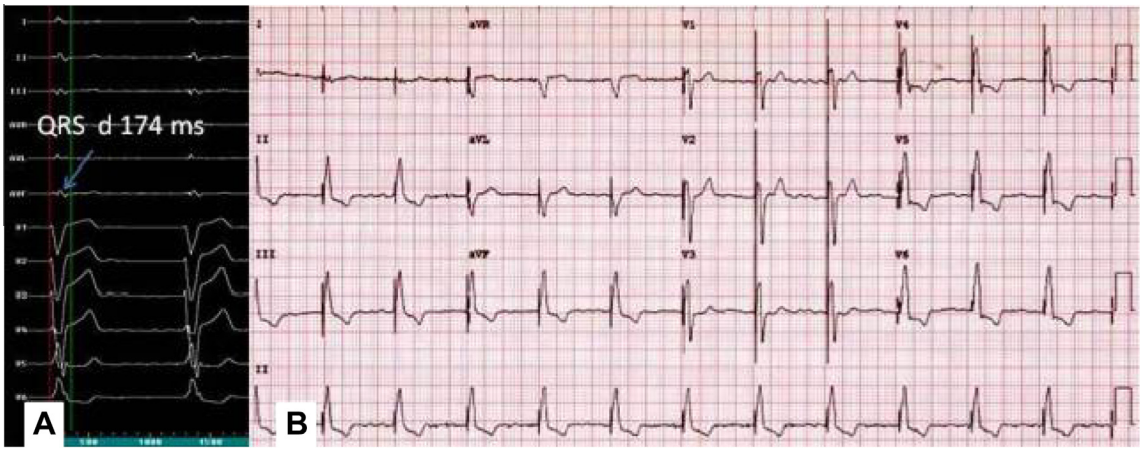

Figure 1. 12 lead ECG tracings; A – Baseline; B – Post CRT-D (QRS d- QRS duration).

lation therapy was optimized as appropriate based on clinical response. The patient was discharged home and was advised cardiac resynchronization therapy with defibrillator implantation. The AF was considered to be permanent given that no details regarding the onset were available and the cardiac diagnosis was left ventricular non-compaction. As such, no other treatable causes for AF, other than the heart-failure, such as thyrotoxicosis were identified. A rate control line of management for atrial fibrillation was considered most appropriate at this stage. The justification for this decision was evident subsequently, when the patient had device-delivered shocks which defibrillated his heart back to an AF rhythm from VF during device implantation. Additionally, review of subsequent inappropriate (due to conducted AF) shock-episode tracings did not demonstrate reversion to a regular rhythm either. The chances of this patient remaining in sinus rhythm after cardioversion, even if that were possible, were considered low. CRT-D implantation was performed on 18/03/2010, about five weeks after initial evaluation at our centre.

Informed consent was obtained from the patient according to standard hospital procedure and the possibility of using the atrial-port for a RV septal pacing lead which is not currently standard practice was fully explained. It was also explained to the patient that in the event of a suspicion of deleterious effects, switching to a VVIR mode would by-pass in entirety the pacing from the RVS in which case standard 'bi-ventricular' VVIR option would be operative. The CRT-D procedure was performed after withholding oral anticoagulant therapy under cover of twice daily sub-cutaneous Enoxaparine, using conscious sedation/prophylactic antibiotics/local anaesthetic infiltration. A left sub-clavicular approach was used for a cephalic vein cut-down and this permitted manipulating a Medtronic Sprint Quattro 65 cm active fixation RV shock lead to the right ventricular apex. Subclavian vein punctures were used for the second right ventricular lead-a 58 cm active fixation Medtronic 4076 lead positioned to pace high in the basal right ventricular septum and for the left ventricular lead-a 88 cm 4196 Medtronic bipolar lead positioned in a posterolateral tributary of the coronary sinus close to the left ventricular basal region (see Fig. 2). The pacing parameters were acceptable and a Medtronic Maximo II CRT-D device was connected to the leads at the appropriate ports with the exception of the RV septal pacing lead which was connected to the atrial port.

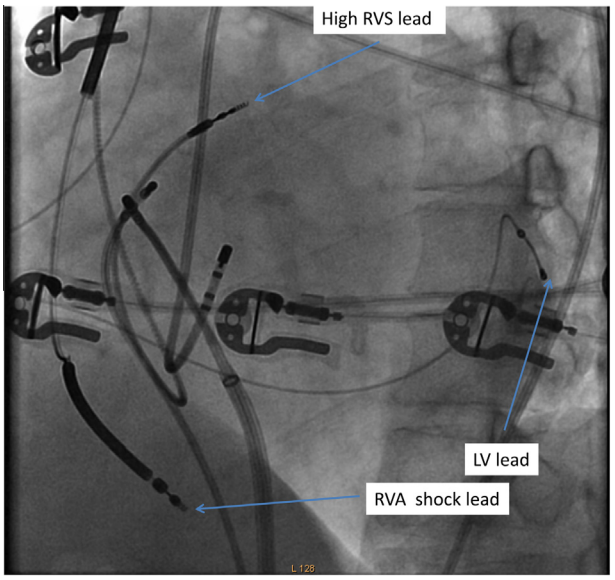

Figure 2. LAO view during AV node ablation procedure by retrograde aortic approach; high RVS, RVA and LV lead orientation.

Defibrillation testing was performed with a successful 15 J shock delivered through the device defibrillating the heart back to previous atrial fibrillation rhythm with a shock impedance of 52 Ohms and good sensing at a sensitivity setting of 1.5 mV. The device was programmed to a modified multi-site CRT (i.e., DDDR, which essentially is multi-site 'ventricular pacing only' mode in this context, lower rate 70 beats per minute, upper sensor and tracking rate 130 beats per minute) 30/20 ms RV septum to LV to RV apex delays (since the RV septal lead was connected to the atrial port, this is shown as AV delay by the device) and mode switch was turned 'OFF' for the same reason. Both sensed and paced 'A'–V delays were programmed at 30 ms. After the initial RVS/LV/RVA pacing amplitude @3.5/4/4 V, respectively (0.4 ms pulse width) immediately post-implant, it was subsequently programmed to 1/4/3 V, respectively (0.4 ms pulse width) based on capture threshold measurements. Although no diaphragmatic capture was evident at implant procedure, later on, an increase in LV capture threshold was noted with diaphragmatic stimulation and hence the LV lead pacing configuration was changed from LV tip to ring and LV tip to RV (A) coil was programmed. Three zones for therapies were programmed (fast VT zone via VF) 'ON' with ATP during charge for VF/before shocks for cardioversion in the VT and FVT zones and all lead impedance measurements were found to be in the acceptable range. PR Logic based algorithms were programmed 'OFF'. 'Onset' as enhancement was left 'ON'.

The patient had to be brought back to the hospital for multiple inappropriate shocks delivered through device for conducted rapid AF (highly variable R–R interval seen in both the RVS and RVA channels with no suggestion of T-wave over-sensing) probably precipitated by increased physical activity level as a result of decreased symptoms. The patient was re-admitted for Atrio-ventricular node ablation to minimize conducted AF. This was successfully performed by retrograde Aortic approach as it was unsuccessful using the venous approach from the right-side of the ventricular septum following which the ventricular pacing increased from 80% to 100%. The 12-lead surface ECG showed a QRS duration of 126 ms with positive deflections in the inferior leads and a negative deflection in the avL lead and a good transition on the anterior chest leads suggesting good CRT pacing result (see Fig. 1). A subsequent ECG and Pulsed Wave Tissue Doppler guided assessment of degree of inter and intra-ventricular delays in different pacing modes demonstrated a clearly shorter surface ECG QRS duration in the LV + 30 ms RVA and RVS + 30 ms LV + 20 ms RVA pacing modes compared with RVS or RVA alone (see Table 1) [3]. Similarly, once again, a clearly shorter surface ECG QRS onset to Pulsed Wave Tissue Doppler myocardial systolic velocity onset intervals suggesting better intra and inter-ventricular delays based on the Pulsed Wave Tissue Doppler derived pre-ejection period were noted in these modes and the LV + RVA mode was slightly better of the two. Maximum inter-ventricular delay occurred in the RVS only mode and in the RVA only mode, all the intervals measured were very prolonged but with much less intra and inter-ventricular dyssynchrony. Since the improvement in the patient occurred in the RVS to +30 ms LV to +20 ms RVA mode, the basic parameters were not altered after the Pulsed Wave Tissue Doppler assessment of optimal cardiac resynchronization parameters except for increasing the LV to RVA delay to 30 ms. Conducted AF response and V-rate stabilization was turned 'OFF' after atrio-ventricular node ablation. V-sense response and PVC response was left

Table 1. Distribution of ECG and ultrasound pulsed tissue Doppler based QRSD and segmental pulsed tissue Doppler myocardial velocity onset delays by pacing modes.

	Intervals (ms) measured based on pacing site(s)			
	RVS + 30 ms LV + 20 ms RVA (BiV DDD* mode)	RVS alone (AAI mode)	RVA alone (VVI mode)	LV + 30 ms RVA (BiV VVI mode)
Surface ECG QRS duration	173	197	254	169
QRS onset to LV basal septum $S_{m\ onset}$	151	197	208	144
QRS onset to basal lateral LV $S_{m\ onset}$	155	165	190	148
QRS onset to basal anterior LV $S_{m\ onset}$	141	141	204	158
QRS onset to basal inferior LV $S_{m\ onset}$	151	183	232	158
QRS onset to basal lateral RV $S_{m\ onset}$	141	130	197	155

$S_{m\ onset}$ – Pulsed Wave Tissue Doppler Myocardial Systolic Velocity onset; LV – Left Ventricle; RV – Right Ventricle; RVS – RV septum; RVA – RV apex; BiV – Biventricular pacing.

* Selection of DDD mode in this situation implies multi-site ventricular pacing in RVS → LV → RVA sequence.

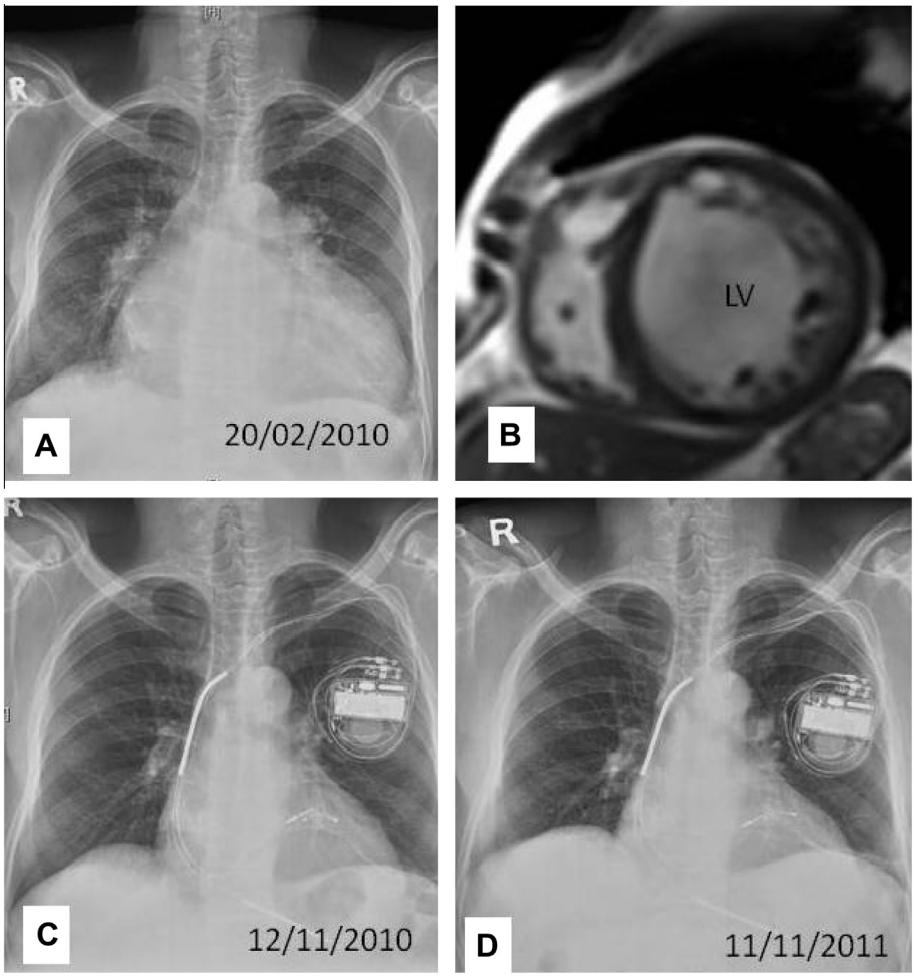

Figure 3. A & B – Baseline chest X ray and MR image; C & D – Serial follow up chest X ray images.

'ON'. The patient showed significant progressive improvement in effort tolerance and was asymptomatic at last check one and a half years post implantation. Serial chest radiographs showed progressive decrease in the cardiac size (see Fig. 3) and echocardiography showed significant improvement in the overall left ventricular function to almost near normal with a LVIDD/IDS of 44/32 mm, however, with persisting features of left ventricular non-compaction.

Discussion

This case report demonstrates the benefits of optimal medical management combined with proven technology. In addition, a novel use for the atrial-port in this patient with atrial fibrillation where it would otherwise have been left plugged. The patient showed a good CRT result as evidenced by a narrower QRS on surface ECG and the clinical response notwithstanding the very limited options available in the various ventricular pacing permutations possible because of the programming features of the atrial channel which in this situation is used as a ventricular channel. There is no way of proving in this case that the benefit seen in the form of clinical response to treatment accrues from anything beyond cardiac resynchronization therapy in a general sense and it definitely is not possible to attribute any benefit to the RVS-first pacing mode. In this case report the patient may well have improved significantly due to just optimal medical management, adequate ventricular rate control for AF and conventional CRT-D because all of these modalities have incremental value in the management of such patients. Whether or not using the atrial port in this fashion is beneficial remains to be evaluated by further studies. If the device industry takes up the challenge of better programmability of 'A'–V delays and V–V delays to accommodate this form of multi-site pacing, more room for finer

adjustments would be possible post implant procedure (such as simultaneous or LV to RV septum with or without ventricular apex pacing).

Conclusion

This case report showcases the possibility of non-conventional sequential multi-site pacing (RV septum to LV to RV apex) being feasible using the standard CRT-D device in patients with atrial fibrillation where the atrial port would otherwise be left plugged.

References

[1] Wells G, Parkash R, Healey JS, Talajic M, Arnold JM, Sullivan S, et al.. Cardiac resynchronization therapy: a meta-analysis of randomized controlled trials. CMAJ 2011;183(4):421–9.
[2] Khadjooi K, Foley PW, Chalil S, Anthony J, Smith REA, Frenneaux MP, et al.. Long-term effects of cardiac resynchronization therapy in patients with atrial fibrillation. Heart 2008;94(7):879–83.
[3] Maurizio Galderisi, Fabio Cattaneo, Sergio Mondillo. Doppler echocardiography and myocardial dyssynchrony: a practical update of old and new ultrasound technologies. Cardiovascular Ultrasound 2007;5:28.